avoided with aspirin, which effectively equates these complications with coronary events. While imperfect, this adjustment is a reasonable approximation and shows successfully that aspirin is the most cost effective treatment despite complications. Indeed, by adjusting for complications only for aspirin, Marshall has been conservative: clopidogrel increases the incidence of rash and diarrhoea,[13] and statins in primary prevention have not consistently reduced the incidence of myocardial infarction or stroke and have not reduced all cause mortality, possibly because of undetected serious adverse events.[14]

Should these results persuade clinicians? Should national guidelines be amended to offer preventive measures in order of incremental cost effectiveness? Absolutely, because any other action guarantees less gain in health for whatever is spent. Across the entire NHS, following the current guidelines would waste billions of pounds and prevent fewer coronary events than if cost effectiveness were used to guide treatment. Evidence based clinical guidance must include incremental cost effectiveness, to prevent the pointless and profligate pursuit of perfection.

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Competing interests: None declared.

1 Berwick DM. Is it wise to promote "perfection" as a goal? *Continuous Improvement* 2003;(4). www.ihi.org/pursuingperfection/faqs/ci0601letter.asp (accessed 11 Nov 2003).
2 Stock SR, Gafni A, Bloch RF. Universal precautions to prevent HIV transmission to health care workers: an economic analysis. *CMAJ* 1990;142:937-46. (Inflation indexed and converted from published 1990 estimate of C$8m to C$129m by RNW.)
3 Department of Health. *Building a safer NHS for patients*. London: DoH, 2001.
4 National Quality Forum. *Safe practices for better healthcare*. Washington, DC: NQF, 2003.
5 Standing Medical Advisory Committee. *The use of statins*. London: Department of Health, 1997.
6 Freemantle N, Barbour R, Johnson R, Marchment M, Kennedy A. The use of statins: a case of misleading priorities? *BMJ* 1997;315:826-8.
7 Sackett DL, Richardson WS, Rosenberg WMC, Haynes RB. *Evidence-based medicine: how to practice and teach EBM*. New York: Churchill Livingstone, 1997.
8 Marshall T. Coronary heart disease prevention: insights from modelling incremental cost effectiveness. *BMJ* 2003;327:1264-7.
9 Neuhauser D, Lewicki AM. What do we gain from the sixth stool guaiac? *N Engl J Med* 1975;293:226-8.
10 Department of Health. *National service framework for coronary heart disease*. London: DoH, 2000.
11 Heart Protection Study Collaborative Group. MRC/BHF Heart protection study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomized placebo-controlled trial. *Lancet* 2002;360:7-22.
12 Gaspoz JM, Coxson PG, Goldman PA, Williams LW, Kuntz KM, Hunink MG, et al. Cost effectiveness of aspirin, clopidogrel, or both for secondary prevention of coronary heart disease. *N Engl J Med* 2002;346:1800-6.
13 Hankey GJ, Sudlow CL, Dunbabin DW. Thienopyridine derivatives (ticlopidine, clopidogrel) versus aspirin for preventing stroke and other serious vascular events in high vascular risk patients. *Cochrane Database Syst Rev* 2000;2:CD001246.
14 Therapeutics Initiative. Do statins have a role in primary prevention? *Ther Lett* 2003;48:1-2. www.ti.ubc.ca/pages/letter48.htm (accessed 11 Nov 2003).

Risk factor scoring for coronary heart disease

Prediction algorithms need regular updating

Global risk assessment has become an accepted component of clinical guidelines and recommendations in cardiovascular medicine. The aim is to provide a valid estimate of the probability of a defined cardiovascular event over a period of five or ten years in individuals free of clinical manifestations of cardiovascular disease at the time of examination. The information available for global risk assessments commonly consists of individual risk factor measurements and a basic assessment of concurrent clinical conditions. The aim of the resulting absolute level of predicted risk is to determine the intensity of clinical intervention. What do we know about the validity of the population data from which the individual risk factor measurements are derived?

The Framingham Heart Study and the Framingham Offspring Study were the first epidemiological studies that prospectively collected population based data on the association between risk factors and the occurrence of fatal and non-fatal coronary and other cardiovascular events in a systematic and sustained fashion.[1] Hence, when the New Zealand Guidelines Group first used global cardiovascular risk assessment as a tool for identifying patients in need of antihypertensive drug treatment,[2] risk equations based on the experience of the Framingham sample were the only accurate data source readily available. Others followed the approach of using absolute, rather than relative, risk estimates as clinical treatment decision aids, and within a couple of years the Framingham risk equations had pervaded most clinical guidelines.

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Early reports provided reassurance by confirming that observed and predicted risk were of similar magnitude, for example in UK patients.[3] More recent comparisons revealed reasonable agreement between Framingham predicted risk and observed risk in six US cohorts of white and black people, but not in those of Japanese, Hispanic, or Native American ethnic origin.[4] The Framingham authors themselves had cautioned about generalising from their data.[1] And, indeed, an increasing number of reports suggest that this procedure is misleading under various circumstances. When applied to different populations, for example from Southern Europe,[5 6] or in studies with a more recent onset and follow up period,[7 8] the observed absolute risk is often substantially lower than predicted by the Framingham algorithms.

In this issue (p 1267), Brindle et al present their findings for men who participated in the 10 year follow up of the British Regional Heart Study.[9] They report that the Framingham prediction equations overestimate the risk of coronary mortality by 47% and of fatal plus non-fatal coronary events by 57%. Likewise, a recent report from the PRIME study group

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confirmed overestimation by 34% in a male sample from Belfast.[10]

Several reasons account for this overestimation of absolute risk. Firstly, the Framingham baseline assessment was performed in 1968-75.[1] Declining secular trends in cardiovascular mortality and morbidity, as shown impressively in the MONICA project,[11] account for a widening gap between predictions based on disease rates observed in the past and event rates obtained in more recent study periods. Secondly, populations differ substantially in their absolute cardiovascular risk levels,[11] implicitly limiting the external validity of any prediction algorithm that is based solely on one population. Thirdly, increasing proportions of the population are treated with blood pressure and lipid lowering drugs, so attenuating the predictive power of a given untreated risk factor level at baseline. Finally, population specific levels and trends in potentially interacting risk factors, such as alcohol consumption, homocysteine, or triglycerides, may further confound absolute risk predictions.

Brindle et al discuss the many adverse implications that overestimation of risk may have on informed decision making by doctors and patients, on appropriate allocation of healthcare resources, and on public health strategies. To overcome this problem in their study, they used a simple recalibration method by multiplying individual predicted risk with the average ratio of observed over predicted risk. This approach assumes roughly constant ratios across age, sex, and regional groups, and there is no external validation. More general recalibration methods have been suggested before that seem to work effectively in different settings.[4 6] However, they require valid data about mean risk factor levels and survival in a population. Another approach was put forward by the SCORE study group.[12] These investigators pooled data from several cohorts from European countries with high and low cardiovascular mortality levels in order to derive common risk functions. Charts were produced that can be applied to patients from European high and low risk populations. When assessed in independent population cohorts these charts performed reasonably well.[12]

The assessment of absolute risk is currently accepted as a potentially attractive clinical decision aid. What it takes to foster confidence in its application, however, is up to date epidemiological data—collected in surveys, registers, and, when possible, cohorts from populations with varying risk levels—that can be used regularly to adapt prediction algorithms.

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Competing interests: None declared.

1 Anderson KM, Wilson PW, Odell PM, Kannel WB. An updated coronary risk profile. A statement for health professionals. *Circulation* 1991;83:356-62.
2 Jackson R, Barham P, Bills J, Birch T, McLennan L, MacMahon S, et al. Management of raised blood pressure in New Zealand: a discussion document. *BMJ* 1993;307:107-10.
3 Ramachandran S, French JM, Vanderpump MPJ, Croft P, Neary RH. Using the Framingham model to predict heart disease in the United Kingdom: retrospective study. *BMJ* 2000;320:676-7.
4 D'Agostino RB, Grundy S, Sullivan LM, Wilson PW. Validation of the Framingham coronary heart disease prediction scores. Results from a multiple ethnic groups investigation. *JAMA* 2001;286:180-7.
5 Menotti A, Puddu PE, and Lanti M. Comparison of the Framingham risk function-based coronary chart with risk function from an Italian population study. *Eur Heart J* 2000;21:365-70.
6 Marrugat J, D'Agostino R, Sullivan L, Elosua R, Wilson P, Ordovas J, et al. An adaptation of the Framingham coronary heart disease risk function to European Mediterranean areas. *J Epidemiol Comm Health* 2003;57:634-8.
7 Hense HW, Schulte H, Lowel H, Assmann G, Keil U. Framingham risk function overestimates risk of coronary heart disease in men and women from Germany—results from the MONICA Augsburg and the PROCAM cohorts. *Eur Heart J* 2003;24:937-45.
8 Thomsen TF, McGee DM, Davidsen M, Jorgensen T. A cross-validation of risk scores for coronary heart disease mortality based on data from the Glostrup Population Studies and Framingham Heart Study. *Int J Epidemiol* 2002;31:817-22.
9 Brindle P, Emberson J, Lampe F, Walker M, Whincup P, Fahey T, et al. Predictive accuracy of the Framingham coronary risk score in British men: prospective cohort study. *BMJ* 2003;327:1267-70.
10 Empana JP, Ducimetiere P, Arveiler D, Ferrieres J, Evans A, Ruidavets JB, et al. Are the Framingham and PROCAM coronary heart disease risk functions applicable to different European populations? *Eur Heart J* 2003;24:1903-11.
11 Tunstall-Pedoe H, Kuulasmaa K, Mähönen M, Tolonen H, Ruokokoski E, Amouyel P, et al. Contribution of trends in survival and coronary-event rates to changes in coronary heart disease mortality: 10-year results from 37 WHO MONICA Project populations. *Lancet* 1999;353:1547-57.
12 Conroy RM, Pyorala K, Fitzgerald AP, Sans S, Menotti A, De Backer G, et al. Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project. *Eur Heart J* 2003;24:987-1003.

Is the NHS getting better or worse?

We need better data to answer the question

See News p 1250

The NHS is a shambles, and you are too much of a coward to say so. This was the gist of an email I received from an NHS consultant a few weeks ago. I answered—weakly in his eyes—that I couldn't be sure that the NHS was collapsing. I met many people who agreed with him but also many who thought otherwise. I didn't see clear evidence. Yet whether the NHS is improving may be the most important political question in Britain. The government, which has increased NHS expenditure by billions and launched into a 10 year modernisation plan, insists that it is improving. The opposition alleges that the money is being wasted. The people want a better health service, and a billion pound investment that came to nothing would be a national tragedy. So what is the answer? The main conclusion of an extensive, independent review funded by the Nuffield Trust and published this week is that we don't have the data to answer the question reliably.[1] This in itself is an indictment—particularly when the NHS is awash with bodies auditing and inspecting it.

The review—which is of quality in the NHS in England not the other three home countries—has been conducted by Sheila Leatherman, an American professor with appointments in both the United States and the United Kingdom, and Kim Sutherland from the Judge Institute in Cambridge. They describe the review

BMJ 2003;327:1239–41